Fenner (C. S.)

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RESUMÉ OF THE MODERN VIEWS

OF THE

Pathology and Treatment of Glaucoma

By C. S. FENNER, M. D.

LOUISVILLE, KY.

READ BEFORE THE KENTUCKY STATE MEDICAL SOCIETY, 1874.

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PATHOLOGY AND TREATMENT OF GLAUCOMA.

Previous to the invention of the ophthalmoscope but little was known of the pathology of glaucoma. From the greenish reflex of the fundus of the eye it derived the name of glaucoma or green cataract. Authors took much pains to point out the distinguishing symptoms between the incipiency of this disease and the first stages of cataract and amaurosis. Mackenzie says, "To distinguish glaucoma from cataract, especially in the incipient stage, proves to beginners one of the most difficult pieces of diagnosis, and sometimes not to beginners only, but to those who for a length of time have attended to the diseases of the eye. . . . Glaucoma is often mistaken for amaurosis, from the circumstance of being always attended by some of the subjective symptoms of this disease."

When it was sufficiently developed to make the diagnosis clear and unmistakable the patient had to be assured that he would sooner or later become totally blind, and no hope would then remain for the restoration of his vision. Now what a change! By the aid of the ophthalmoscope the first stages of the disease can be diagnosticated with almost unerring certainty; the pathological changes watched as they progress; and, thanks to the genius of Von Graefe, we are enabled to assure the suffering patient that his sight can with great probability be entirely preserved, or at the worst will be but slightly impaired. Thousands are now in the enjoyment of good vision who had they lived a quarter of a century ago would have been hopelessly blind.

Mackenzie first pointed out the fact that an increased hardness of the eye always existed in glaucoma; but he failed to discover the causes of the increased intra-ocular tension, and of its effects on the interior structure of the eye. Even after the monocular ophthalmoscope came into general use it was some time before surgeons learned

to correctly interpret all the phenomena the interior of the eye presented. In the examination made with but one eye of the observer the effect of stereoscopic vision with both eyes was wanting; depressions seemed as elevations, and the glaucomatous cupping of the optic disk appeared and was described as an arching forward of the nerve-entrance.

Von Graefe first detected this error, and showed that what appeared as an elevation was an optical illusion; that the disk was actually pressed backward, forming an excavation. From this change in the form of the optic papilla, taken in connection with the increase of the intra-ocular tension, he saw at once that new light was thrown on this hitherto obscure disease. He argued that the cupping of the optic disk was the result of the increased intra-capsular tension. It is precisely the same as if we were to take a hollow ball of some slightly elastic substance and force into it a fluid. The more thrown in the harder the ball becomes. Now if a small portion of its walls is weaker than at other parts, this weaker or thinner portion, offering less resistance to the internal pressure, would give way and be forced outward. This is just what takes place in glaucoma. There is an increase of the fluid contents of the capsule; that part of the sclerotica corresponding to the entrance of the optic nerve and blood-vessels (lamina cribrosa) is perforated to allow the vessels and nerve-fibers to pass into the eye; around these perforations the sclerotica is thicker and stronger than at any other place, because here it receives the addition of the outer sheath of the optic nerve; hence the edge of the scleral foramen is sharp, very firm, and unyielding. The fibers of the scleral tissue passing between the nerves and blood-vessels, offering less resistance to the increased intra-ocular pressure, being the weaker part, gives way and is pressed backward. As they pass behind the thickened ring surrounding the lamina cribrosa they not only expand backward, but laterally, forming a flask-shaped sack, having an opening corresponding to the resisting edges of the scleral perforation. The nerve-fibers and blood-vessels are put upon a stretch and bent over this sharp edge. If an elastic tube conveying fluid is bent over the sharp edge of a hard substance, the walls of the tube are compressed at that point, and the passage of fluid in them is more or less impeded. Now this is just what takes place in the blood-vessels accompanying the optic nerve. They are bent and stretched over the

resisting edge of the scleral foramen; the walls of the arteries, being firmer and more resisting than the walls of the veins, still continue to convey the blood, but in less quantities than normal; the veins being thinner and more compressible, the circulation in them is impeded to a greater degree; hence they become engorged and swollen. The obstruction and lateral pressure exerted on the arteries prevents their gradual yielding to the impulse of the blood, and they only receive the blood after the impulse has become very strong; hence the arterial pulsation is seen spontaneous and jerking. The capsule being distended by the intra-ocular pressure of the fluids of the eye until it has lost its elasticity, each arterial pulsation adds to the incompressible fluid contents of the globe. The result is that each arterial pulsation increases suddenly the lateral pressure on the veins, and forces by quick compression venous blood through the obstruction at the edge of the optic papilla, while they immediately refill, to go through the same process after each pulsation of the arteries. Thus we have spontaneous arterial pulsation and venous pulsation, which is pathognomonic of increased tension of the eye in glaucoma. By pressure on the normal eye spontaneous arterial pulsation and pulsation in the veins can easily be produced. Bearing these points in mind, most of the phenomena connected with glaucoma are easily explained, and are just what *à priori* would be expected: certain results follow certain causes. We should look for just such pathological changes from increased intra-ocular tension or great rigidity of the sclerotica; and if means could be found to restore the eye to its normal tension, the loss of vision would be prevented or restored if organic lesions beyond reparation had not already taken place.

This is what Graefe accomplished. He found means to relieve this increased intra-ocular tension and restore the eye to its normal hardness, and thus to check those pathological changes that surely tend to organic lesions, causing total loss of vision. If he had done nothing else tending to the advancement of ophthalmological science, his name would be imperishable. He found by experiment that an iridectomy, removing a large section of the iris up to its peripheral attachment, relieved the increased intra-ocular tension, and re-established the normal endosmotic equilibrium between the circulation in the blood-vessels and the fluid contents of the eye.

When he first published his views on this subject to the world in

1856 his conclusions were received with a good deal of incredulity, because this happy result of the operation could not be explained on rational principles. Many surgeons repeated the operation without deriving the expected benefit. I remember seeing a case of glaucoma in the clinique of Desmarres, in 1860, in which he had a short time previously removed a portion of the iris. The vision of the patient was not improved. It was a case of confirmed glaucoma, where, according to our present knowledge, no benefit could be expected beyond relief from pain, because organic changes had taken place beyond the power of reparation. As surgeons became more familiar with the use of the ophthalmoscope, and learned more correctly to diagnosticate the earlier symptoms of glaucoma, and to appreciate the effects of increased tension of the eye, they were led to perform the operation of iridectomy at an earlier period, according to the instructions of Von Graefe, with results so favorable that it is now an almost universally established operation for the relief of increased intra-ocular tension.

Symptoms of Glaucoma.—"These comprehend the senile increase of the hardness of the globe, the ophthalmoscopic appearances of excavations of the optic nerve, the symptoms of hyperæmia and inflammation, as well as a great variety of disturbances of vision."—(*Stellwag.*)

An abnormal hardness of the eye is one of the first objective symptoms, and this increased tension varying from a slight degree, as evidenced by the touch, to the hardness of bone or ivory. A rapid diminution of the refractive power of the eye is usually noticed (*presbyopia*), which the patient is at first disposed to regard as the result of senile changes, and he seeks relief in convex glasses. These have frequently to be changed for those of higher power, until he finds that he requires their use in order distinctly to see remote objects (*hypermetropia*). This diminution in the refractive power of the eye is probably owing to the intra-ocular pressure, which has a tendency to cause the cornea to assume a spherical shape corresponding to the curvature of the sclerotica. In the latter stages of glaucoma the ridge around the sclero-corneal junction disappears. The increase of intra-ocular pressure may be rapid, or it may be very slow and only noticed at certain times, as after a full meal, excesses in drinking, loss of sleep, etc. The premonitory attacks are most usually periodical, and may

be attended by intense headache and ciliary neuralgia, with evidence of acute inflammation; or the disease may steadily progress to total loss of vision without pain or perceptible inflammatory symptoms. The increased tension is evidently due to an excess of fluid secretion in the vitreous. As the eye grows more and more hard the pupil becomes widely dilated, the iris motionless, and forms a narrow ring around the margin of the cornea; it together with the crystalline lens is pushed forward till they rest against the posterior surface of the cornea, and thus entirely obliterate the anterior chamber. The secretion of the aqueous humor ceases from atrophy of the uveal portion of the anterior chamber. The cornea becomes anæsthetic, varying in degree according to the amount of intra-ocular tension: this insensibility may be evenly distributed over its entire surface or confined to certain portions of it. When the internal pressure becomes very great and continues for a length of time, the cornea becomes so insensible that its surface may be rubbed with the finger or brushed with a feather without causing any pain or reflex action. This insensibility is undoubtedly due to a pressure on or a stretching of the nerve-fibers supplying this part of the globe. Irritating substances lodge upon the surface of the cornea, and as they produce no pain or reflex action are not rubbed off by nictation or washed away by the tears; hence they remain there, giving rise to inflammation, ulceration, opacities, etc.—results similar to those following paralysis or injuries of some of the branches of the fifth pair of nerves. Greatly enlarged veins finally are seen bursting out from the anterior portions of the sclerotica, which anastomose and pass tortuously over the equator of the globe, while near the sclero-corneal junction there is a fine vascular net-work of vessels, more particularly evident during an attack of intense inflammation. Finally the sclerotica becomes thin, translucent, and of a whitish porcelain appearance. As the hardness of the eye increases the disturbances of sight become more and more manifest. The field of vision is gradually obscured. In daylight a thick fog prevents distinct perception of objects; by artificial light, as in looking at a lighted candle in a dark place, a halo is seen around the flame that gives all of the colors of the rainbow, the inner side being red, the outer green. Soon objects cease to be perceived on the inner part of the visual field; this insensibility gradually extends eccentrically above and below until the ends meet on the outer side, leaving a

narrow slit of sensitive retina extending downward and outward. This gradually closes, until finally all quantitative perception of light is gone. The patient long after this has periodically subjective sensations of light; hence he has his light and dark days.

Ophthalmoscopic Appearances.—When the increased hardness of the eye becomes plainly manifest the optic disk is found slightly excavated or pushed backward; the arteries are of normal size or slightly diminished in caliber; the veins are swollen and a little flattened. As the intra-ocular pressure increases the disk becomes more and more cupped, the arteries diminish in size, while the veins are larger and more tortuous. The blood-vessels passing over the edge of the disk are seen bent or arched backward; venous pulsation becomes visible, or may be produced by slight pressure with the finger, followed by spontaneous arterial pulsation. When the tension of the eye becomes very great the excavation of the disk is deep and flask-shaped; all its blood-vessels disappear; its color, which at first, owing to venous congestion, was of a reddish hue, now becomes of a grayish-blue or a tendonous-white. The retinal vessels are sharply cut off at the edge of the disk, where they appear beak-shaped, particularly those which pass in an oblique direction. The veins are greatly enlarged, and the collateral branches assume a corkscrew appearance, while the arteries are more or less atrophied. Retinal ecchymosis is often seen. In the advanced stages of glaucoma a characteristic yellowish-white ring surrounds the optic-nerve entrance, which increases in breadth as the excavation grows deeper.

The above-enumerated symptoms and pathological changes constitute glaucoma, which may or may not be accompanied by inflammation; hence the disease is usually described under the heads of inflammatory and non-inflammatory glaucoma.

Inflammatory Glaucoma.—This form of the disease is generally attended by a premonitory stage, each attack being followed by a perfect intermission, which may last for weeks or months; the attacks grow more and more frequent, until after a time there ceases to be a perfect intermission, but only a remission of the more acute symptoms. The glaucoma is now said to be confirmed. During the permonitory stages the patient when looking at a lighted candle sees around the flame the prismatic halo. This appearance may be at first the result of simple pressure on the nerve-fibers, but after a time it is generally

accompanied by a cloudiness of the aqueous and vitreous humors; hence the prismatic halo and haziness is probably due to diffraction. This haziness is at first transitory, sometimes appearing several times during the day, and lasting but a few minutes. When the inflammatory symptoms become more severe the blueish-gray haziness is so great and the fundus of the eye is so completely obscured that an ophthalmoscopic examination can not be made. The premonitory attacks are generally preceded or accompanied by severe headache, with ciliary neuralgia. If the inflammation is of very high degree, these pains are often agonizing, and vision may be totally lost in the course of a few hours (*glaucoma fulminans*); but generally this result does not take place until after repeated exacerbations, each one of which adds to the impairment of vision, until at the end all quantitative perception of light disappears. The haziness that obscures the field of vision during the premonitory stages often disappears after sleep. In persons advanced in life the eye gives from its fundus a greenish reflex; but this is not seen before middle age. Senile changes in the lens give its nucleus a yellowish-amber color. In glaucoma the bluish-white opacity of the vitreous mingling its color with the amber-colored lens gives a greenish reflex. The green color disappears if the lens is removed, showing that it is only a combination color.

Chronic Inflammatory Glaucoma.—This form of the disease is also accompanied by a premonitory stage, but usually there is a remission of the more prominent symptoms, rather than perfect intermission. The intra-ocular tension slowly increases; but the pain is less severe than in the acute form, or it may be entirely absent. The progress of the disease is slow; but the final result is the same—total loss of vision. Frequently before this takes place there are one or more attacks of acute exacerbation. These exacerbations may continue long after vision is totally destroyed, giving rise to intense pains in the head, with severe ciliary neuralgia; the inflammation may extend to the conjunctiva, causing chemosis and great swelling of the lids.

Non-inflammatory Glaucoma (*Glaucoma Simplex*).—This form of the disease is insidious in its approach, and often makes considerable advances before the patient is aware of it. If confined to one eye, he often accidentally finds that he can not see as well with it as with a sound one. Small objects are indistinctly seen, and the limitation

of accommodation progresses to hypermetropia. This limitation of accommodation can at first be remedied by convex glasses. There is no prismatic halo around a lighted candle; but after a while vision becomes misty, and objects are indistinctly perceived. The eye has a normal appearance, but the iris is a little sluggish in its movements. To the touch, the globe will be found to be a little harder than natural, and the ophthalmoscope reveals a slight displacement of the porus opticus backward, with perhaps a bending of the retinal vessels in the optic papilla, and by slight pressure venous pulsation and spontaneous arterial pulsation can be produced.

The disease may remain almost stationary for months or years, but eventually the intra-ocular tension slowly increases, the iris dilates to a narrow ring, the aqueous chamber diminishes in size, the eccentric contraction of the field of vision from the inner side gradually extends, the cornea becomes anæsthetic, the optic disk deeply cupped, and the eye approaches the hardness of bone. All of the pathological changes enumerated as resulting from increased intra-ocular tension take place, until finally all perception of light is gone, often without the patient having suffered pain or the appearance of inflammatory symptoms. There is an absence of retinal ecchymosis in such cases; but more frequently before vision is totally destroyed exacerbations of acute inflammation come on and hasten the final result.

Secondary Glaucoma is where the increase of intra-ocular tension supervenes or results from some other affection of the eye, as large, deep cicatrices of the cornea, serous choroiditis, anterior or posterior synechia or irido-cyclitis. The latter is often induced by wounds of the anterior capsule, whereby the aqueous humor is admitted to the lens, causing the latter to swell from the absorption of the fluid, and thus to press on the iris and ciliary body. This is why it is so dangerous to freely open the anterior capsule in the operation of discission for cataract in persons advanced in life. The irritation induced by a hard lens displaced in the vitreous humor, causing irido-cyclitis, terminating in glaucoma, is the chief reason why so many eyes have been lost a few months after the performance of the now-abandoned operation of couching.

Treatment.—In the premonitory stage of glaucoma, when a considerable time intervenes between the attacks, great care should be exercised to avoid every thing that has a tendency to induce hyper-

æmia of the retina or choroid. The diet should be nutritious, but the stomach not overloaded with indigestible food. Hearty suppers and all excesses in drinking spirituous liquors should be avoided. The habits should be regular, particularly as to the hour of retiring, so as to avoid loss of sleep. Care should be taken not to use the eyes too long in continued reading fine print, writing, etc., and particularly to avoid reading in a recumbent position. If there is a diminution in the powers of accommodation, convex glasses should he worn of sufficient power to make objects distinctly seen without straining the eyes.

The chief object of treatment is to restore the eye to its normal tension. This was formerly attempted to be accomplished by evacuating a portion of the vitreous, but without producing the desired result. Next the frequent tapping of the anterior chamber was tried. But the benefits derived from these operations were only temporary, few cases of glaucoma having been permanently relieved by this course of treatment. The only effectual remedy is an iridectomy carefully performed according to the instructions given by Von Graefe. It is best to remove the portion of excised iris upward, for then the upper lid covers the coloboma, and prevents circles of diffusion that would result from a removal of other parts of the iris. The incision should be made in the sclerotica at least half a line from the corneal junction. While an excision of a portion of the sphincter of the iris, as in the operation for artificial pupil, may in many cases give relief; yet to obtain the full benefits of the iridectomy at least one fifth of the iris should be removed and torn completely from its peripheral attachment. Great care should be taken that no portion of the iris remains in the angle of the wound.

Graefe says, "Although it is certainly advisable to excise the iris as peripherally as possible, yet the non-fulfillment of these conditions is by no means always, not indeed usually, followed by insuccess. I have seen a very great number of eyes in which the operation had been imperfectly performed for typical inflammatory glaucoma, and yet in which the cure had been permanent. . . . I would fain hope that these remarks will not induce carelessness in excising the iris." He considers it of paramount importance "that the iris should be carefully and completely removed from the wound, *for any inclusion of it in the cicatrix will certainly increase the secretory irritability of the eye.* Had I to choose between a coloboma that does not reach quite to the

peripheral wound, but in which one edge of the coloboma is largely included in the cicatrix, I should always prefer the former. . . . . *In no case should the operation be concluded till we are certain that the iris is completely released from the wound.*"

In simple non-inflammatory glaucoma a little delay in performing the operation is a matter of no great importance; but in the acute form of the disease iridectomy should be performed as speedily as possible. If done within the first fourteen days of the attack, we may "restore and preserve to the eye nearly or entirely its full former functionary power."—(*Stellwag.*)

In chronic inflammatory glaucoma it not infrequently happens that the patient is not seen by the surgeon until serious lesions have taken place. An iridectomy may arrest the progress of the disease, but often fails to completely restore the eye to its normal functions, because organic lesions have taken place beyond the power of reparation. Usually a few days after the operation the eye assumes very nearly or quite its normal hardness; the anterior chamber refills with aqueous humor; the cupped disk is flatter; and the pathological changes become stationary or diminish.

It sometimes happens that the iridectomy does not check the glaucomatous process. In such cases Graefe recommends a supplementary iridectomy, incising a diametrically opposite piece of the iris. He says, "I must indeed confess that I have become dubious whether incision of the adjacent piece has any influence over the tension. It is at all events very slight, and will generally not suffice in cases where the first iridectomy has had no sufficient or permanent effect. On the contrary, I could adduce at least a dozen cases where excision of the opposite piece has had a permanent effect after failure of the first iridectomy."

There is a singular result frequently following an iridectomy in acute inflammatory glaucoma. It is this: if the disease is confined to one eye, the operation, in a great majority of cases, hastens the appearance of the disease in the other eye; it generally comes on in from eight to fourteen days after an iridectomy on the glaucomatous eye. The patient should be apprised of this beforehand, and assured that a similar operation will with great probability soon be required on the other eye.

It is always best to place the patient thoroughly under the influence

of chloroform previous to performing the iridectomy, as the evacuation of the aqueous humor suddenly relieves the eye of its great tension, and there is then a tendency in the degenerate walls of the blood-vessels to give way and permit intra-ocular hemorrhage. The risk is greatly increased by the spasmodic contraction of the ocular muscles during the operation. These spasms are prevented in a great measure by rendering the patient insensible by means of an anæsthetic.

It has not yet been satisfactorily explained how an iridectomy diminishes the increased or abnormal intra-ocular tension of the eye. Wells says, "Amongst other hypotheses, some have thought that the tension was diminished by the excision of a considerable portion of the secreting surface; others that the removal of the iris quite up to its ciliary insertion, and the consequent exposure of the zonula zinnii, facilitates the interchange of fluid between the vitreous and aqueous humors, and thus diminishes the difference in the degree of tension between these humors. We must admit, however, that this problem has not at present been satisfactorily solved."

Graefe, in the last article he published on glaucoma, says, "No progress of importance has been made in respect to the *theory* of the operation, notwithstanding many attempts. Old hypotheses are usually dragged forth, little as they avail for the explanation of existing facts. The unhappiest of all—the one referring the effect simply to the wound of the ocular capsule—is periodically brought forward, decked in some new formula, and recommended for adoption. Fortunately for our patients, practice advances independently on the foundation of empirical principles. No doubt some day a satisfactory theory will be found; but in the mean time we must not be led away by transient hypotheses."